Angiographic-Morphologic Correlation in Patients with Severe Mitral Regurgitation Due to Prolapse of the Posterior Mitral Valve Leaflet


SUMMARY

Angiographic-morphologic correlation was done on findings in 16 patients with proven prolapse of the posterior mitral leaflet and severe mitral regurgitation. A triscalloped bulge into the left atrium in the left ventriculogram taken in the right anterior oblique projection is caused by prolapse of all three scallops of the posterior mitral leaflet. The prolapsed middle scallop produces a central bulge, the prolapsed posteromedial commissural scallop a posteroinferior bulge in the area of the posterior commissure, and the prolapsed anterolateral commissural scallop an anterosuperior bulge in the anterolateral commissural area. The latter is often overlapped by the aortic root but if prominent, juts beyond it. Correlation proved that the diagnosis of individual scallop prolapse in this syndrome can be made accurately despite gross mitral regurgitation.

Additional Indexing Words:
Mitral click syndrome
Ruptured chordae tendineae

Prolapse of the posterior leaflet of the mitral valve (PPML), causing regurgitation as well as life-threatening arrhythmias and sudden death, has been the subject of a number of recent reports. The spectrum of clinical signs in this syndrome varies from the presence of a nonejection systolic click with or without a systolic murmur, to the presence of severe mitral regurgitation with a holosystolic murmur and no click. Electrocardiographic findings characterized by ST and T wave changes in the inferior leads have been observed in some patients.

Left ventricular cineangiograms are used to confirm the PPML, but angiographic-morphologic correlations have not been emphasized. Pertinent to any consideration of the angiographic diagnosis is a recent redefinition of the morphology of the normal mitral valve. This study demonstrated that the posterior leaflet was a triscalloped structure in 92% of normal hearts with a large middle scallop (MS) and two smaller commissural scallops, one on either side of it. The latter were named respectively anterolateral and posteromedial commissural scallops (ALCS and PMCS). Prolapse of any part of this triscalloped structure should be recognizable in a left ventricular cineangiogram taken in the right anterior oblique (RAO) projection. In previous papers we demonstrated the angiographic appearance of a pathologically-proven MS prolapse and suggested that prolapse of a commissural scallop should produce a bulge at either extremity of the posterior leaflet. The present study was undertaken to establish the angiographic appearances produced by prolapsed scallops of the posterior mitral leaflet and to determine the accuracy of angiographic diagnosis of individual scallop prolapse in this syndrome.
Materials and Methods

Sixteen patients with PPML confirmed either at surgery or at autopsy were studied. The left ventricular cineangiograms taken in the RAO projection were available for review in all cases. The extent, location, and outline of the prolapsed scallops of the posterior mitral valve leaflet, as they bulged into the left atrium in systole, were carefully identified.

The morphology of the 16 mitral valves was studied by pinning them onto a waxed board and carefully defining the parts of the leaflet that were diseased and the nature of that disease. The gross changes were recorded and photographs made.

The angiographic and morphologic studies were done by two sets of observers, neither of whom had prior knowledge of the cases. A committee was made and then the results were correlated comparing the presence or absence of angiographically identified scallop prolapse with the presence or absence of morphologic evidence of prolapse.

Results

Clinical and Hemodynamic Findings in Patients with PPML

There were nine males and seven females in the group studied. Their ages ranged from 37 to 69 years. All had isolated mitral regurgitation with an apical holosystolic murmur and left ventricular enlargement. The murmur was Grade IV-V/VI in 14 individuals. All patients were significantly disabled with class III-IV symptoms of heart failure (New York Heart Association classification). Ten had ECG findings of left ventricular hypertrophy while 11 had atrial fibrillation. None of the patients had clinical or electrocardiographic evidence of ischemic heart disease. Gross mitral regurgitation was demonstrated angiographically in each person. In 12 the hemodynamic findings reflected the severe degree of mitral regurgitation.

Angiographic Findings in PPML

The mitral valve plane lies along the atrioventricular ring at the base of the opacified left ventricle. The anterior and posterior leaflets overlap each other in the RAO projection. The normal mitral valve shows very little systolic bulging beyond the atrioventricular ring. A smooth, doming effect may be present, but never a distinct bulge.

In contrast, patients with PPML have distinct convex bulges into the left atrium in systole (figs. 1 and 2). The prolapsed MS produces a central bulge. The aortic root may overlap the anterolateral half of this bulge, but the overlap is minimal when the left ventriculogram is taken with optimal obliquity (30°). A bulge in the area of the posteromedial commissure is caused by prolapese of PMCS (figs. 1 and 2). This bulge, situated at the postero inferior extremity of the posterior leaflet, may be oval or have a distinctive “eagle beak” configuration (fig. 3). The prolapsed ALCS, which produces an anterolateral bulge in the anterolateral commissural area (figs. 1 and 2), is often overlapped by the aortic root. However, when the bulge is prominent, it juts beyond the aortic root.

A triscallop prolapse of the posterior leaflet was observed angiographically in four patients; a biscallop prolapse in six and single scallop prolapse in the remaining six. MS prolapse was seen most frequently (15 of 16 patients). It occurred either as a single scallop prolapse (five patients) or in combination with prolapse of other scallops (ten patients). Isolated prolapse of a commissural scallop occurred only once. It involved the PMCS. ALCS involvement occurred only as part of a triscallop prolapse. When biscallop prolapse was noted it always involved the MS and PMCS (six
patients). Thus, of the 48 scallops examined by cineangiographic assessment in the 16 patients, 30 were thought to have prolapsed.

**Morphological Observations**

Each of the 16 patients had a triscalloped posterior mitral valve leaflet. Of 48 scallops examined, 27 were abnormal, hooded, and bulged toward the left atrium (figs. 2, 4, and 5). These prolapsed scallops were thickened (figs. 2, 4, and 5) and had a gelatinous appearance on cut section. Histologically, most showed myxomatous degeneration and were thickened by connective tissue. The chordae tendineae attached to the prolapsed scallop tended to be elongated and thickened (fig. 4). Some showed focal myxomatous changes. Ruptured chordae were associated with prolapsed scallops in seven of the valves (figs. 4 and 5 and table 1). In six patients the ruptured chordae had been attached to a prolapsed MS (fig. 4).

The anterior leaflet showed myxomatous degeneration in five of 16 patients; but it was not severe. None of the leaflets showed morphological changes compatible with its having prolapsed, and none had ruptured chordae tendineae attached to it.

**Angiographic-Morphological Correlation**

Of the 48 scallops studied, 30 showed prolapse on angiography and 27 showed morphological changes compatible with the prolapsed state. Thus, angiographic-morphologic correlation was correct in 90% of prolapsed scallops of the posterior mitral valve leaflet. Indeed, prolapse of either the MS or the ALCS was always identified correctly. In three
patients, angiography indicated a prolapsed PMCS. However, morphological observations did not demonstrate a typical bulged, hooded appearance on this scallop.

Discussion

In the literature the description of the angiographic appearance produced by PPML varies from paper to paper. We believe that this stems in part from a misconception that the anterior mitral valve leaflet has a wider attachment to the atrioventricular annulus than the posterior leaflet, whereas the opposite is true. In addition, it has not been appreciated that the posterior mitral valve leaflet is usually a triscalloped structure. Again, angiographic-morphologic correlations were done in only one presentation. The present study provides such correlations in 16 patients with PPML and permitted an accurate definition of the angiographic appearances produced by this condition. Thus, prolapse of the MS produces a central bulge of the posterior leaflet into the left atrium while bulges resulting from prolapse of commissural scallops are situated at the posteroinferior and anterosuperior extremities of the posterior leaflet.

Although they did not provide angiographic-morphologic correlations, Criley et al. deduced that a shadow in the “postero-lateral or inferior margin of the atrio-ventricular ring” was caused by PPML. Jeresaty correctly interpreted the bulges produced by prolapsed MS and PMCS but did not prove this morphologically. He also related a bulge in the anterosuperior half of the mitral valve to a prolapsed anterior leaflet. We disagree with this latter interpretation. Our observations indicate that a bulge in this location is due to prolapse of the
ALCS rather than to prolapse of the anterior leaflet.

Each of the 16 patients studied had marked functional disability from severe isolated mitral regurgitation confirmed by hemodynamic investigation and by ventriculography. However, the clinical findings did not help define the cause of the mitral regurgitation. Left ventricular angiograms proved essential to detect the presence of prolapse of part or all of the posterior mitral valve leaflet. Nevertheless, in spite of gross mitral regurgitation, the angiographic diagnosis of individual scallop prolapse was proven accurate in 90% of instances.

Acknowledgments

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References

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Figure 5

Surgically-excised mitral valve from a patient with angiographically identified prolapse of PMCS. Note thickened, hooded PMCS. A black cord is attached to it at the site of a ruptured chordae tendineae. The other scallops of the posterior leaflet are normal. The posteromedial half of the AL is thickened slightly. Scale indicates 1 cm.
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